

EXPLANATION OF THE BILATERAL DEFICIT IN HUMAN VERTICAL JUMPING

Maarten F. Bobbert, Wendy W. de Graaf and Jan N. Jonk

Institute for Fundamental and Clinical Human Movement Sciences, Vrije Universiteit, Amsterdam, The Netherlands;
email: M_F_bobbert@fbw.vu.nl

INTRODUCTION

In the literature, it has been reported that in two-leg jumps, humans achieve less than twice the jump height and work they are able to achieve in a one-leg jump [1, 2]. The seemingly submaximal performance in two-leg jumps has been coined bilateral deficit. It has been speculated that the deficit is primarily caused by a reduction in neural drive to the muscles in the two-leg jump and only secondarily by differences in non-neural factors [1, 2]. The purpose of the present study was to investigate the contribution of differences in active state and shortening velocity to the bilateral deficit in vertical squat jumping. For this purpose, we performed experiments on human subjects and simulations with a forward dynamic model of the musculoskeletal system.

METHODS

Eight male subjects performed maximum height vertical jumps, pushing off with only their right leg or with both legs together. In the one-leg jumps, the left leg was kept as passively as possible. Except for the position of the left leg, the initial body configuration was the same in both types of jumps (Figure 1). Kinematics and ground reaction forces of the individual legs were collected at 200 Hz, and EMG activity was recorded at 1000 Hz from soleus, gastrocnemius, vasti, rectus femoris, glutei and hamstrings of the right leg. Off-line, the EMG signals were high-pass filtered, rectified and low-pass filtered at 5 Hz to produce SREMG (Smoothed Rectified EMG). Inverse dynamics was used to calculate work produced by the right and left leg during the push-off.

One- and two-leg jumps were also simulated with a forward dynamic model of the musculoskeletal system [3]. The model had 7 rigid segments (foot, shank and thigh of each of the two legs and a head-arms-trunk segment), and was actuated by 15 muscle-tendon complexes (MTC): soleus, gastrocnemius, vasti, rectus femoris, glutei and hamstrings of the left and right leg, and tibialis anterior, short head of biceps femoris and iliopsoas of the left leg. Each MTC was represented as a Hill-type unit. The model calculated the segmental motions corresponding to stimulation (STIM) input to the muscles. Initial STIM levels were chosen such that static equilibrium was achieved in the starting position, which was close to that of the subjects. Subsequently STIM of each muscle was allowed to switch between "on" and "off" several times, and switching times were optimized to find maximum jump height using a parallel genetic algorithm. Once the optimal solutions had been found for the one-leg and two-leg jumps, we used them to determine the cause for differences in muscle work performed. We estimated the effect of differences in active state by substituting at each MTC length in the two-leg jump the active state at the same MTC length in the one-leg jump, and subsequently re-calculating muscle force and work (this step will be called: correction for active state). The same procedure was followed to estimate the effect of differences in CE velocity (correction for CE-velocity).

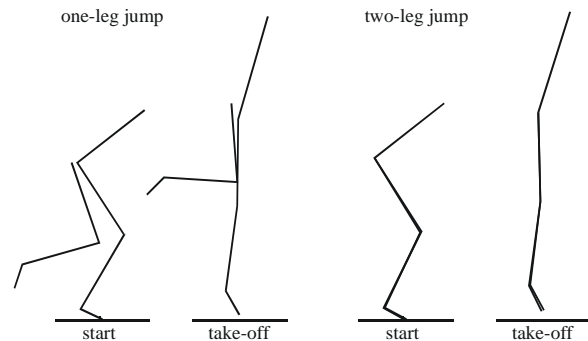


Figure 1: Average initial and take-off body configurations of the subjects in the one-leg and two-leg jumps.

RESULTS AND DISCUSSION

No differences occurred between the two types of jumps in the range over which the subjects extended the joints of their right leg (Figure 1). Obviously, in the two-leg jump, greater velocities were reached and the range of motion was traveled in less time than in the one-leg jump. Mean vertical displacement in the airborne phase was 23.7 cm in the two-leg jump and 12.6 cm in the one-leg jump. Work performed by the right leg was 195 J in the two-leg jump and 248 J in the one-leg jump. Ratio's of peak SREMG values in the two-leg jump to peak SREMG values in the one-leg jump, averaged over all subjects, ranged from 0.9 to 1.0 for the different muscles, suggesting that any reduction of neural drive in the two-leg jump was at best minimal. In the simulation model, the work performed by the right leg was 160 J in the two-leg jump and 234 J in the one-leg jump. In the model, the difference in work output of the right leg was, of course, completely caused by non-neural factors. Correction for active state caused the work in the two-leg jump to increase by 24 J, while correction for CE-velocity caused it to increase by 58 J. This shows that the differences in active state and shortening velocity between two-leg jumps and one-leg jumps can have a substantial effect on the force and work output of the muscles.

CONCLUSIONS

SREMG-results indicate that at best a minimal reduction in neural drive occurred in two-leg jumps compared to one-leg jumps. However, in a two-leg jump, initial active state is lower than in a one-leg jump and less time is available to increase active state. Moreover, the muscles travel their range of shortening at greater speed. In the model, these two factors combined caused the muscles to produce substantially less work in the two-leg jump than in the one-leg jump. It seems that the bilateral deficit in jumping is primarily caused by non-neural factors rather than by a reduction in neural drive.

REFERENCES

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